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CONTENTS

Symposium on Systemic Aspects of Periodontal Diseases

Introduction and Conclusions . BALINT ORBAN . . . 123

Gingivitis In Pregnancy . . . A. W. MAIER . . . 123

A Study of the Histopathology of the Gingiva in Patients With Diabetes Mellitus . . . . . HAROLD G. RAY . . . 128

Roentgenographic and Bacteriologic Aspects of Periodontal Changes in Diabetics . . . . . S. SIEGMUND STAHL . . . 130

Histopathologic Changes in the Gingiva in Leukemia . . . FRANK M. WENTZ . . . 132

Home Care of the Mouth

I. *Proper Home Care of the Mouth* W. J. CHARTERS . . 136

II. *Home Care Technics—Ancient and Modern* . . . . . DOROTHEA F. RADUSCH . 138

III. *Teaching Home Care to the Patient* . . . . . DICKSON G. BELL . . . 140

Editorials 144

Precocious Advanced Alveolar Atrophy SAMUEL CHARLES MILLER . 146

Nomenclature in Periodontia 147

Nutrition and Dental Health . . . DOROTHEA F. RADUSCH . 150

Book Review 153

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The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society.

Ninth Postgraduate Seminar

THE AMERICAN ACADEMY OF PERIODONTOLOGY

May 2, 3, 4, 1949

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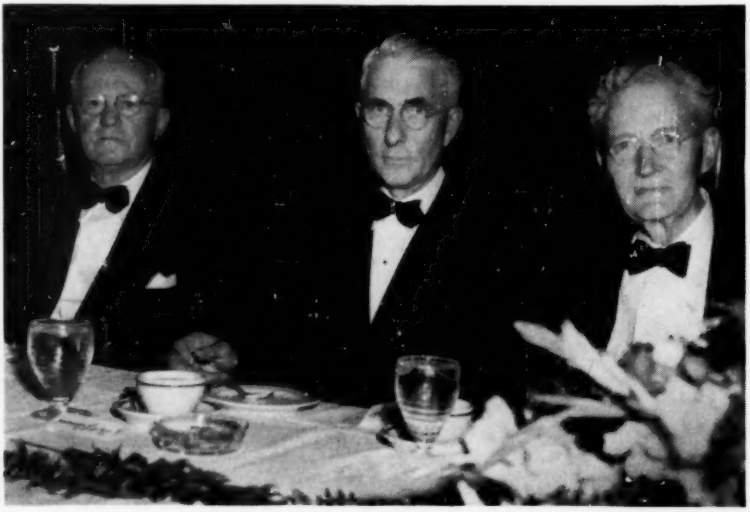
CODE OF ETHICS

American Academy of Periodontology

Section I

In his dealings with his patients and in all his relations with the members of his profession, the dentist should be governed by the Golden Rule: "All things whatsoever ye would that men should do to you, do ye even so to them." There is no higher Code of Ethics.

Fellow Certificates awarded to
Drs. Carlos H. Schott, Harold J. Leonard, and Edgar D. Coolidge
at Academy Banquet in Chicago, September 10, 1948



THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FORTY-EIGHT

Symposium on Systemic Aspects of Periodontal Diseases*

By A. W. MAIER, M.D., D.D.S.
HAROLD G. RAY, D.D.S.
S. SIEGMUND STAHL, D.D.S., M.S.
and FRANK M. WENTZ, D.D.S., M.S.

Introduction and Conclusions

By BALINT ORBAN, M.D., D.D.S.

IT HAS often been claimed that systemic disturbances are etiologic factors in the development of periodontal diseases, but their effect in the production of periodontal lesions has never been made clear. In an attempt to solve this problem we have investigated, during the past two years, three different systemic disturbances, associated in dental literature with periodontal diseases: pregnancy, diabetes, and leukemia.

We wish to acknowledge the coöperation of Drs. Isaac Schour and Maury Massler.

Gingivitis In Pregnancy†

By A. W. MAIER, M.D., D.D.S. **

This investigation has been conducted in an attempt to expand our present knowledge and clarify our interpretation of two important problems. *First:* To what extent are we justified in correlating systemic disturbances with gingival lesions? Since pregnancy, although a physiologic state of systemic imbalance, is known in literature to be connected with gingival lesions, gingivitis in pregnancy has been chosen for this investigation, especially because the clinical picture had not been very well correlated with the histologic findings.

Second: Are there any clinical and histopathologic changes in the gingiva which are exclusively characteristic of pregnancy?

Time does not permit me to present a detailed review of the vast amount of literature available on this subject. I shall confine myself to mentioning those

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These investigations were supported (in part) by a research grant from the division of Research Grants and Fellowships of the National Institute of Health, United States Public Health Service.
Presented at the Thirty-second Annual Meeting of the American Academy of Periodontology, September 10, 1948, *Chicago, Illinois.*

†This paper will be published *in extenso* in the *Journal of Oral Surgery, Oral Pathology and Oral Medicine.*
**[Dr. Maier was in charge of the investigation of gingival disturbances in pregnant women. He reports on the clinical and histopathologic findings.]

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authors whose work and research have been outstanding and extensive in the field of gingivitis in pregnancy, such as Monash, Merritt, Hirschfeld, Burket, Blum, and especially Ziskin and his co-workers.

There are various reports in literature concerning the prevalence of gingivitis in pregnancy:

Biro stated that 38% of pregnant women are affected by gingivitis. Freund, Pinard and Schmidt, reported that 53% to 60% of pregnant women showed gingivitis while, in control studies, 15% to 18% of the same number of non-pregnant women showed gingivitis. Looby (as quoted by Burket) reported that the examination of 475 cases of pregnant women revealed 52% affected by gingivitis. Ziskin stated that the examination of 416 cases of pregnant women showed 37.9% affected by various degrees of pregnancy gingivitis.

The present study is based upon the examination of 530 pregnant women between the ages of 16 to 40 years and ranging from the third to the ninth month of pregnancy. The patients were members of a low income group and 50 per cent belonged to the Negro race. Mouth hygiene was fairly good, only very few cases showed gross neglect.

The clinical examination was carried out with the aid of mouth-mirror and flashlight and was concerned mainly with the anterior region of the oral cavity. The condition of the gingiva was recorded on a "Gingival Diagnostic Chart" which was designed for the purpose of thoroughly describing clinical features and considering important diagnostic aspects. This table shows the clinical features of the normal gingiva in pregnancy as well as those of mild, moderate, and severe gingivitis. The importance of this chart should be stressed because by following its sequence the clinician is able to observe and describe systematically the clinical features of the gingiva.

In the absence of an available standard index, an attempt was made to classify the degree of gingival inflammatory reaction according to its progressive changes into five groups.

Group 1. No Pathology	236 or 44.6%
Group 2. Mild Inflammation	190 or 35.9%
Group 3. Moderate Inflammation	93 or 17.5%
Group 4. Severe Inflammation	8 or 1.5%
Group 5. Tumor Formation	3 or 0.5%

Some patients were selected for biopsy investigation and, in each case, color photographs were taken. These cases ranged from those with clinically normal appearing gingiva to those with severe hyperplastic changes. Fifty-three biopsy specimens were obtained.

Group 1. THE NORMAL GINGIVA

Clinical Appearance: The normal gingiva is characterized by the pink color, normal contour of the gingival margin, pointed papillary contour, stippled texture, and firm consistency of the attached gingiva. There is no exudate present and the gingival sulcus is extremely shallow. (This description follows the sequence shown in the Gingival Diagnostic Chart). The free gingiva is separated from the attached gingiva by the free gingival groove and the attached gingiva is separated from the oral mucosa by the muco-gingival junction*.

Histologic Analysis: The surface of the epithelium of the attached gingiva often appears to be wavy. Parakeratotic cells cover the surface. Epithelial proliferation is evidenced by the irregular outline of the epithelial ridges and the presence of pegs. A slight indentation on the surface corresponds to the free gingival groove, while other depressions and elevations represent the stippling of the attached gingiva. Numerous mitotic figures can be observed in the stratum germinativum. The surface of the free gingival sulcus usually shows no hornification; it is continuous and without ulceration.

The connective tissue of the lamina propria in the attached gingiva consists of collagenous bundles and relatively few cellular elements. In the reticular layer the collagenous bundles are heavier and there is no inflammatory cell accumulation or edema. The collagenous bundles become thinner in the free gingiva; the tissue here is usually more cellular and, frequently, slight inflammatory reactions might be observed near the bottom of the gingival sulcus. The inflammatory cells consist of lymphocytes and plasma cells. A few scattered polymorphonuclear leukocytes can be seen, some in the

*For terminology See Orban: "Clinical and Histologic Study of the Surface Characteristics of the Gingiva." *J. Oral Surg., Oral Med. and Oral Path.* 1:827, 1948.

PREGNANCY		NORMAL	MILD GINGIVITIS	MODER. GINGIVITIS	SEVERE GINGIVITIS
Color	Pink	+	+	+	
	Red		++	+++	+++
	Magenta			+	+++
	Pale Pink				
Marginal Contour	Normal	+			
	Swollen		+	++	+++
	Receded				
	Irregular				+++
Papillary Contour	Pointed	+			
	Blunted		+	++	+++
	Flat				++
	Inverted				
Texture	Stippled	+	+	+	
	Glossy			++	+++
	Pebbly				
	Granular				
Consistency	Firm	+		+	
	Turgid		+	+++	
	Boggy				+++
	Dense				
Exudate	None	+	+		
	Serous		+	+	+++
	Purulent			+	+
	Hemorrhagic				++
Pockets	None	+			
	Shallow	+	+	+	
	Moderate		+	+	+
	Deep				++

connective tissue, some of them migrating through the epithelium.

Group 2. MILD GINGIVITIS

Clinical Appearance: In these cases the color of the gingiva in general is pink, but usually the tips of a few papillae show slight redness. The gingival contour is mostly normal, the interdental papillae pointed, but those which show increased redness are slightly swollen and blunted. A sharp line of demarcation can be seen between these papillae and the attached gingiva; their texture is frequently somewhat granular. The attached gingiva is stippled, the consistency is slightly turgid. No exudate is present and pockets are extremely shallow.

Histologic Analysis: Generally a microscopic examination reveals a somewhat advanced inflammatory condition. The epithelium of the attached gingiva is usually very regular and the surface is covered by a hornified layer. The epithelial ridges are of equal length and width and there are very slight surface indentations indicating stippling. The free gingival groove, separating the free from the attached gingiva, is frequently accentuated and deep. The epithelium of the free gingiva usually shows a slight and irregular parakeratotic layer, the stratum spinosum extensive hydropic degeneration, and the stratum germinativum numerous mitotic figures. Heavy and wide epithelial ridges give this epithelium a very irregular appearance. Approaching the pocket side of the gingiva, microabscesses often appear in the surface layer of the epithelium. The pocket epithelium becomes very thin in certain areas and is invaded by polymorphonuclear leukocytes. The irregular epithelial proliferations in this region usually show numerous mitotic figures.

The lamina propria of the attached gingiva consists of collagenous bundles without any pathologic manifestation; the capillaries in this region are normal. The lamina propria of the free gingiva is frequently edematous and slightly infiltrated with inflammatory elements. The inflammatory exudate usually becomes very heavy in the reticular layer, especially as the gingival pocket is approached. The inflammatory cell elements consist mainly of plasma cells, some lymphocytes, and polymorphonuclear leukocytes in the pocket area. Between the plasma cells, areas of necrosis can be observed. The capillaries are dilated and the endothelium is swollen. The number of capillaries is increased and they seem to form numerous branches.

Group 3. MODERATE GINGIVITIS

Clinical Appearance: Generally, the color of the free and attached gingiva is pink. However, there usually is a definite tendency toward increased redness in several of the interdental papillae. The gingival margin in these areas is slightly swollen. The papillae are fairly well pointed except those which show increased redness; they are swollen and blunted, often increased in size. Slight, but distinct, lines of demarcation can be observed between free and attached gingiva of the unaffected papillae, while they fade and disappear in those which are swollen and increased in size. The texture of the gingiva is glossy, the consistency is highly turgid. Usually a slight serous exudate can be observed and pockets are alternatingly shallow and moderately deep.

Histologic Analysis: The surface of the epithelium is usually wavy in appearance, it forms heavy and irregular ridges and shows severe hydropic degeneration in the stratum spinosum. There is a slight flattening of the surface cells in some regions, but hornification is seldom observed. Pyknosis might be evident close to the surface. Usually there is no sign of a free gingival groove. Frequently hyaline degeneration and accumulation of hyaline droplets is evident at the margin of the gingiva. The epithelium in the pocket becomes extremely thin in certain areas and shows proliferation of pegs into the underlying inflamed connective tissue. In certain areas the epithelial covering is often completely missing, exposing a highly vascular and heavily infiltrated granulation tissue. The pocket side of the epithelium reveals in most cases numerous microabscesses, containing an abundance of polymorphonuclear leukocytes.

The papillary layer of the connective tissue is in most cases severely edematous; some regions are slightly, others are heavily infiltrated with inflammatory cells, other regions might show only capillaries and fibrous tissue rich in fluid. The reticular layer is usually entirely filled with groups of plasma cells and lymphocytes. The endothelial cells of the capillaries and small vessels are swollen, often almost round and

bulge into the lumen. A very unusual finding is the frequent occurrence of multinucleated (giant) cells in the endothelium. These cells often bulge into the lumen of the vessel, they occasionally spread to the outside and sometimes appear as a solid cord of endothelial cells. Necrosis between plasma cells is slight.

Group 4. SEVERE GINGIVITIS

Clinical Appearance: In these cases the color of the gingiva ranges from magenta to cyanotic-purple with bright red margins in some regions. The gingival contour usually is highly irregular and swollen, the papillary contour rolled, and in some instances inverted. Some of the interdental papillae might be protruding or form mushroom-like growths. The texture of the gingival tissue is highly glossy, the consistency extremely boggy and frequently a hemorrhagic exudate is visible along the free marginal gingiva. Pockets of considerable depth are usually present on all teeth.

Histologic Analysis: In most cases the epithelium appears highly irregular. Numerous epithelial ridges and pegs project into the connective tissue, some showing terminal branches. Hornification of the surface is seldom observed; it is replaced by a thin parakeratotic layer. The stratum spinosum is very irregular, hydropic degeneration is frequent, the basal cell layer is usually thickened and consists of several layers of cuboidal cells with numerous mitotic figures. Toward the pocket area the epithelium frequently becomes more irregular and the epithelial proliferations more pronounced. Its surface is frequently broken, thus exposing connective tissue. These ulcers are covered by a dense fibrinous network interspersed with blood cells, leukocytes and proliferating connective tissue, forming a pseudomembranous covering on the ulcerated surface. The epithelial islands and strands in these areas are usually infiltrated with numerous leukocytes. The superficial blood vessels are thrombosed by a fibrinous clot, and the vessels which are not thrombosed are frequently surrounded by a heavy fibrinous exudate. In the ulcerated areas the inflammation is acute, but is chronic in the deeper layers where usually a dense infiltration of lymphocytes and plasma cells can be observed. In the areas of acute inflammation, the endothelial cells are swollen and numerous leukocytes are present in the lumen as well as in the surrounding tissue. The endothelial cells often show proliferative tendency, characterized by mitotic figures in the vessel walls. The entire connective tissue is mostly edematous and infiltrated with inflammatory cells to such an extent that there sometimes remains only a very limited amount of stroma.

Group 5. TUMOR FORMATION*

Clinical Appearance: These tumors are characterized by their cyanotic-magenta color. At the periphery the tissue often appears highly red, due to irritation and trauma; frequently necrotic ulceration is evident on the surface of the tumor facing the tooth. Hemorrhage to a very slight traumatization is a common feature. Some of the tumors might originate from a broad base, others are pedunculated.

Histologic Analysis: In microscopic detail, these pregnancy tumors do not materially differ from the picture already described in the various forms of gingivitis in pregnancy. There is only an accentuation of the proliferative conditions.

SUMMARY AND CONCLUSIONS

1. The gingival condition of 530 pregnant women between the ages of 16 to 40 years and ranging from the third to the ninth month of pregnancy is reported as follows: 236 or 44.6% presented no pathologic changes; 190 or 35.9% showed mild gingivitis; 93 or 17.5%—moderate gingivitis; 8 or 1.5%—severe gingivitis and 3 or 0.5% showed tumor formation. When these data are compared with those of non-pregnant individuals, there is no significant difference in the prevalence of gingivitis in pregnancy and in non-pregnant individuals.†

2. Clinically, a gingivitis in pregnancy is characterized by the localization of the inflammation to a few gingival papillae which show a rather sharp demarcation from the attached gingiva. This sharp demarcation is due to accentuation of the

*We take this opportunity to express our sincere appreciation to Drs. Theodore Blum, M. S. Aisenberg, Rowe Smith, John Svoboda, and Merrill J. Shepro, for furnishing us with their valuable collection of microscopic specimens as well as color photographs of pregnancy tumors for our investigation.

†Reference is made to a recent investigation, conducted by Drs. Schour, Massler and Stahl, who have examined the gingival condition of 250 young non-pregnant females as well as 1000 young male medical and dental students.

free gingival groove and is caused by an inflammatory swelling of the free gingiva. The edges of the gingival margins are often red, fringed, and swollen and appear sharply demarcated from the attached gingiva, which is pale and firm. Hyperplastic papillae tend to show considerable overgrowth and have been erroneously classified as tumor formation. None of these symptoms are infallibly indicative of pregnancy since they may be found in gingivitis of adolescence and other age groups.

3. The histopathologic features consist of a proliferative inflammation characterized by numerous mitotic figures in the epithelium, endothelium and connective tissue. Hyalinization of the epithelium, pearl formation of epithelial pegs, disturbances of hornification, microabscess formation, accumulation of lymphocytes and lymphoblasts, pseudomembranous ulcerative inflammation, are features of this picture. However, none of these features are exclusively characteristic of pregnancy.

4. Gingivitis in pregnancy is an inflammatory condition, most probably of local irritative origin, and modified by the systemic disturbance. Pregnancy can be considered a conditioning factor which influences the character of the inflammatory reaction. It is not to be considered the primary etiologic factor.

A Study of the Histopathology of the Gingiva in Patients With Diabetes Mellitus*

By HAROLD G. RAY, D.D.S.**

INTRODUCTION

THIS STUDY is part of an investigation of periodontal diseases as they occur in patients suffering from diabetes mellitus. Case histories of the general disease and complete oral findings were recorded for patients from the metabolic clinic of the University of Illinois Research and Educational Hospital. Kodachrome pictures of the oral cavity and full mouth roentgenograms were made of 30 of these patients. The roentgenographic survey and bacterial findings will be reported by Dr. S. S. Stahl. From these 30 patients, gingival biopsies were taken and this report is concerned with the histological examination of these specimens.

MATERIAL AND METHODS

The biopsies were taken from individuals ranging in age from 10 to 71 years, and the duration of diabetes was 3 months to 10 years. The patients were classified into three groups according to the control of the disease. Those consistently showing no glycosuria were placed in the group of good control; those showing traces were classified as fair, and those showing considerable glycosuria were considered to be in poor control. The biopsy specimen removed for study was usually an interdental papilla and included part of the attached gingiva. These specimens were immediately fixed in Zenker-formalin; some in 95 per cent alcohol or acetone, imbedded in paraffin and stained with hematoxylin-eosin, Mallory's connective tissue stain, Gomori's or Bauer—Feulgen's glycogen stain. The histological observations were tabulated according to a chart for each section examined.

FINDINGS

In general the oral clinical picture of many of these patients showed a low grade gingivitis with some evidence of development of periodontal pockets. Some of the cases presented, clinically, a rather normal picture with pink gingiva, well stippled and of good contour. None of the patients showed periodontal abscess formation or any other acute symptoms. In 11 of the 30 microscopic specimens definite hornification of the surface epithelium was observed; 18 showed parakeratosis and, in one, the epithelium was unhornified. None of the sections exhibited hyperkeratosis.

In a large number of the specimens, an accumulation of lymphatic inflammatory elements could be observed in the papillary layer of the connective tissue, between the epithelial ridges and surrounding their pegs. The lymphatic elements also invaded the epithelium. There was no evidence of surface ulceration in these regions. Such inflammatory areas were frequently found in these specimens close to the free surface of the gingiva. They were independent and at a distance from the inflammation near the gingival pocket.

In the reticular layer of the gingiva a considerable increase in the elements of

*A preliminary report: This paper will be published *in extenso* at a later date.

**[Dr. Ray was in charge of the histopathologic aspect of the research on diabetes mellitus.]

chronic inflammation was observed, which was much more pronounced than one would expect from the clinical examination. Twenty-four of the 30 biopsies were characterized by a massive accumulation of lymphocytes, plasma cells, and some macrophages. Foci of necrosis, in varying degrees, could be observed in 25 specimens.

Hydropic degeneration of the epithelium, especially the prickle cell layer, was found in 20 cases. In some instances the nuclei of the epithelial cells showed a peculiar hydropic degeneration, leading to vacuolization; the chromatin material was condensed at the nuclear membrane. Such degeneration, in most cases, affected the nuclei in the basal cell layer close to the deep end of the epithelial ridges and pegs.

In most cases the pocket epithelium showed a leucocytic invasion.

Degeneration of bundles of collagenous fibers was a finding in 14 of these specimens. This degeneration was characterized by pyknotic nuclei of the fibroblasts and by a degeneration of the collagenous fibers, probably of mucoid character, which appeared as a granular mass between normally staining bundles of fibers. Such areas of collagenous fiber degeneration occurred throughout the connective tissue, but were found most frequently in the deeper layers.

A thickening and hyalinization of the walls of large and small blood vessels was observed in 14 cases. The structure of the vessel walls was, homogeneous and hyalin-like; sometimes the layers had lost their sharp definition. The endothelial cells were swollen and protruded into the lumen which, in some cases, appeared to be completely obliterated. Fibrinous thrombosis in numerous vessels was found in one case.

In 9 of the specimens, "hyalinoid" or in some instances calcified bodies were observed. These appeared oval, round or irregular, staining light to dark pink, and were not homogeneous but granular. In some of these bodies, amorphous calcification could also be seen. These bodies were either single or arranged in groups; in one specimen they occurred in a rather long row reaching close to the gingival papilla. They appeared as hyalinized, collapsed cylinders with some necrotic material in the center. Foreign body giant cells were found in one specimen that contained these bodies. Near these "hyalinoid" bodies, the collagenous fibers were in a state of degeneration, and, in some regions, it appeared that the bundles of fibers had become hyalinized and possibly calcified.

DISCUSSION

The correlation of these findings with the presence of diabetes is, of course, difficult. Similar findings can probably be seen in other biopsy specimens. However, as a whole, the findings in the 30 biopsies of diabetics seem to have some significance. Of the 30 cases, 13 were in poor control, 11 in fair control, and six well-controlled. The findings of the epithelial invasion by lymphocytes, thickened vessels walls, degenerated collagenous fibers, hyalinized bodies and epithelial invasion by lymphocytes, thickened vessel walls, degenerated collagenous fibers, hyalinized bodies, and epithelial degenerations were all noted in a much higher percentage in patients, with poor or fair control, than in those well-controlled. This becomes more significant if one compares these findings with a similar number of biopsy specimens obtained from non-diabetics in which similar findings were noted only sporadically. An accurate statistical evaluation can not be attempted at this time since the material is not large enough.

One of the oustanding features of these biopsy specimens was the more severe chronic inflammatory reaction, evidenced by the increased number of lymphatic inflammatory elements. This was particularly significant when compared with the clinical picture of a mild, chronic gingivitis. The inflammatory elements were not confined to the region of the pocket, but were found extending far into the deeper layers. In numerous specimens, lymphocytes surrounded and invaded the epithelial ridges without any sign of surface irritation or ulceration.

The chronic character of the inflammatory reaction might be associated with the degeneration of collagenous fibers found in these cases.

It is known that in diabetes there is an increased breakdown of protein. This local tissue breakdown may lead to local acidosis, which might explain the increase in plasma cell and lymphocytic infiltration.

The thickening and hyalinization of the blood vessel walls could also find its explanation in the diabetic state. It is well known that in diabetes, arterio-sclerosis is an early

and common finding, though the exact cause is not quite clear. The changes of the gingival vessels could be explained by this general vascular susceptibility, possibly enhanced locally by the chronic inflammation.

The hyalin bodies which were found frequently in our specimens might be a co-incidental finding, but it is more probable that they are connected with the degeneration of collagenous fibers and hyalinization of the blood vessel walls. Calcified bodies have been described in gingival biopsy specimens, but the frequency of such findings in our present material seems to be more than co-incidental.

SUMMARY AND CONCLUSIONS

1. Gingival biopsies of diabetics show a chronic inflammatory reaction characterized by the accumulation of plasma cells and lymphocytes. The lymphocytes are often localized in the papillary layer and invade the epithelium. This lymphocytic infiltration is independent of the inflammation in the gingival pocket.

2. The surface of the epithelium showed no characteristic pathologic changes. Some hydropic degeneration occurred in the prickle cell layer and intra-nuclear edema in the germinative layer close to the ends of epithelial cells.

3. Degeneration of collagenous fibers was observed in the papillary, as well as reticular, layer of the gingiva.

4. Thickening and hyalinization of blood vessel walls was a frequent occurrence, sometimes leading to their complete obliteration. Fibrinous thrombosis was observed.

5. Hyalin and calcified bodies were present in a large number of specimens.

6. The diabetic state seems to modify the reaction of the gingival tissues, partly by lowering their resistance, partly by inducing an early vascular damage.

7. The diabetic state might be directly responsible for the degeneration of collagenous fibers due to increased protein breakdown.

Roentgenographic and Bacteriologic Aspects of Periodontal Changes in Diabetics

By S. SIEGMUND STAHL, D.D.S., M.S.*

INTRODUCTION

THE ORAL CHANGES that take place in the diabetic patient have long presented a problem to the dental profession. These changes have been studied in relation to caries, surgical procedures, and periodontal disease.

This study is concerned with certain phases of periodontal disease in the diabetic patient, namely: 1) intra-oral roentgenographic changes in the alveolar process and 2) a survey of changes in the oral flora of these patients.

REVIEW OF LITERATURE

Despite many studies, a clear-cut cause and effect relationship between diabetes and periodontal disease has not yet been demonstrated.

Historically, it is of interest to note that oral changes in the diabetic were reported as early as 1862 by Seiffert. He noted the common complaints to be glossodynia (burning tongue), burning mouth, and dryness (xerostomia). These symptoms have become less frequent since the advent of insulin in 1921.

The oral changes most frequently reported are those found in the periodontium. An increase in the severity of periodontal disease in diabetics as compared with non-diabetics has been pointed out by Rosenthal in 1931, Grott in 1943, Lovestedt and Austin in 1943, and others. These changes in the periodontium include 1) gingival disease as reported by Bloch in 1922; Hirschfeld in 1934, and Ziskin in 1944; and 2) alveolar bone changes as reported by Sheppard in 1936, Aleman and Uleia in 1936, Rutledge in 1940, and others. The gingival changes consisted of a proliferation and hyperplasia of the gingivae. The possible relation of this type of gingival disease with a vitamin deficiency, especially in the vitamin B complex, has been pointed out by Rudy and Cohen in 1942. The alveolar bone changes consisted, most frequently, of alveolar crest atrophy.

The oral flora in diabetic patients, to my knowledge, has not been reported. Of interest, is a report by Richardson in 1933 who stated that diabetic whole blood, regardless of blood sugar level, has, in general,

*[As a member of the research group, Dr. Stahl studied the roentgenographic and bacteriologic aspects of periodontal diseases in diabetics and has compiled an extensive bibliography on this problem.]

a weaker bacteriostatic property than does non-diabetic blood.

MATERIAL

This study was conducted at the Research and Educational Hospitals, University of Illinois, and all patients studied were under treatment by the Metabolic Department of the hospital.

1. Gingival disease was assessed in 56 patients, 16 males and 40 females, 14 to 71 years of age.

2. Of these 56 patients, 42 patients (14 males and 28 females, 14 to 55 years of age) were studied for roentgenographic changes by means of full mouth intro-oral roentgenograms.

3. Thirty-eight patients (27 females and 11 males, 14 to 65 years of age) were surveyed for the presence of hemolytic organisms.

The length of known diabetes varied from 1 to 12 years.

METHODS

Blood sugar determinations were made at various intervals 3 months to 3 years.

Glycosuria determinations were made at regular intervals 1 week to 3 months.

1. The *severity* of the diabetic condition was assessed by correlating the blood sugar level, as determined by the Exton Rose Test, with the amount of glycosuria, as determined by the Benedict's Test

Patients were then classified into one of five groups, A-E.

Group A—100 mgm. per cent—infrequent spilling-traces

Group B—120 mgm. per cent = + 1 glycosuria

Group C—160 mgm. per cent = + 2 glycosuria

Group D—200 mgm. per cent = + 3 glycosuria

Group E—200 mgm. per cent = + 4 glycosuria

Gingival Assessment

The assessment of the gingival condition was made by using a) the P-M-A Index as described by Schour and Massler in 1948, and b) a more thorough clinical examination, where recession, pocket depth, amount of calculus, etc., were recorded. The degree of gingivitis was divided into 5 groups, none, mild, moderate, severe, and very severe.

Roentgenographic Assessment

Intra-oral roentgenograms were studied for alveolar crest resorption, absence or interruption of lamina dura, enlarged marrow spaces, and root resorption, and were classified as no change, mild, moderate, severe, and very severe changes.

Bacteriological Survey

Cultures were made on blood agar plates (human blood) taken from smears of the throat and gingiva of the lower anterior teeth. The material from the swabs was inoculated on the surface of the blood agar plate; one plate, divided in half, was used for both throat and gingival swabs.

The inoculum was later streaked by means of a wire loop. After 24 hours incubation, the plates were examined for the following organisms according to typical types of colonies observed:

A. Pin head colonies—presumably streptococcus 1) non-hemolytic strep. (gamma) —2) greening strep. (viridans or pneumococcus) green zone—3) hemolytic strep. (beta)—definite zone of hemolysis.

B. Large, shiny colonies—presumably staphylococcus 1) hemolytic staph.—2) non-hemolytic staph.—

The amount of growth was noted as: O—no growth; G—growth (approximately 5 colonies); C—considerable growth (more than 5 colonies).

FINDINGS

1. Sixty of 134 patients, were edentulous —41 per cent.

Since *complete* tooth loss is usually due to periodontal disease, the above findings may indicate that a severe type of periodontal disease was prevalent among these patients.

2. Changes of blood sugar level were associated with glycosuria in 38 cases. The coefficient of association was found to be .72, indicating that 20 out of 38 patients showed a perfect association, 13 a good association, and 5 a poor association.

Since the cofficient of .72 associates the two most reliable indices used to determine the degree (or severity) of diabetes, this figure represents the reasonable maximum to be expected in associating any factor such as oral disease and diabetes.

3. The severity of periodontal disease, as judged by clinical examination, was associated with the severity of the diabetic state. In 156 patients, the coefficient of association

was .61, indicating that 24 out of 56 patients showed a perfect association, 24 a good association, and 8 a poor association.

4. The association of roentgenographic changes with the diabetic state resulted in a coefficient of correlation of .65, indicating that 17 out of 42 patients had a perfect association, 18 a good association, and 7 a poor association.

The highest association was noted between horizontal alveolar crest atrophy and an increase in the severity of diabetes.

5. Twenty-five out of 38 patients examined showed presence of hemolytic organisms. 10 patients showed pinhead colonies, presumably streptococcus, 9 patients showed 9 large, shiny colonies, presumably staphlococcus, and 6 patients showed both types of colonies. This percentage of hemolytic organisms is rather high, since Massler and Macdonald, and Massler and Blum have shown that less than 12 per cent of normal young adults show the presence of hemolytic streptococcus, and less than 6 per cent show hemolytic staphylococcus.

6. The severity of periodontal changes was associated with the presence of hemolytic organisms. The coefficient of association was found to be .57.

7. No association was found between the presence of hemolytic organisms and the severity of diabetes.

DISCUSSION

The findings show that with an increase in the severity of the diabetic state, an increase in the severity of roentgenographic changes and gingival changes can be noted in most cases. It is of further interest, to note the association of hemolytic growth and periodontal disease in this survey. While no attempt was made to establish the pathogenicity of the organisms, the frequency of hemolytic organisms present might well demonstrate a probable change in the oral flora. This is not surprising, since it is well known that the slightest change in the internal environment (tissue health) results in a change of the bacterial population.

While no specific changes in the periodontium could be linked with diabetes, the metabolic upset caused by this disease does seem to be associated with periodontal changes.

The significance of the values of the coefficient of association established in this study can only be noted when one considers that the two most reliable indices in this study, glycosuria and blood sugar levels, gave a coefficient value of .72.

CONCLUSIONS

1. Sixty out of 134 patients examined were edentulous.

2. Alveolar bone resorption both in amount and severity tended to increase with an increase in the severity of the diabetic condition.

3. Gingival pathology in general showed a similar trend to alveolar resorption.

4. Twenty-five of 38 patients, showed a growth of hemolytic organisms (normal—
Hemolytic streptococcus = 12%
Hemolytic staphylococcus = 3%)

5. The coefficient of association between the amount of hemolytic growth and the gingival state was .57.

6. No significant association was found between an increase in severity of the diabetic state and amount of hemolytic growth present.

Histopathologic Changes in the Gingiva in Leukemia

By FRANK M. WENTZ, D.D.S., M.S.

INTRODUCTION

LEUKEMIA is an invariably fatal disease of the hemopoietic organs, characterized by an overproduction of white blood cells. While the white blood cells normally found in the blood stream are fully matured, in the case of most types of leukemia, immature forms will appear in the circulation.

The leukocytes are generally divided into granulocytes and agranulocytes. The granulocytes have either neutrophilic, eosinophilic or basophilic granules in their cytoplasm. The lymphocytes and monocytes are without granules and therefore are termed agranulocytes. The parent cells of all leukocytes are derived from undifferentiated cells of the reticulo-endothelial system. In normal states the type of cell produced depends on the site of origin; that is, the neutrophil, eosinophil, and basophil leukocytes arise from the reticulum of the red bone marrow, lymphocytes from the reticulum of lymphatic tissue, and monocytes, very probably from the reticulum of the spleen. However, in disease states, the reticulum in any tissue may give rise to these cells.

There are a number of developmental stages between the parent cell of all blood cellular elements and the mature functioning cells which circulate in the blood stream.

The myeloblast is the mother cell of the granulocytes. The granulocytes pass through the stages of premyelocyte, myelocyte, metamyelocyte, and a stage which is called the stab form. In the latter the nucleus does not show the mature lobulation, as does the adult polymorphonuclear leukocyte, but the granules in the cytoplasm are well developed. As previously stated, under normal conditions only, the mature leukocytes are found in the circulating blood, but in cases of myeloid leukemia all developmental stages might be found.

The lymphoblast is the parent cell of the lymphocyte. Only two stages are known between the early and the adult form. These are the large lymphocyte and the medium lymphocyte. Lymphatic leukemia also shows all developmental stages in the blood. The same is true for monocytic leukemia. In this type the monoblast is the mother cell which in turn gives rise to the adult monocyte.

In classifying the leukemias, the prevailing cell is significant. If lymphocytes predominate we speak of a lymphatic leukemia; if the granulocytes are involved, we term the case myeloid leukemia. According to the degree and the acuteness of the disease, leukemias are classified as acute, subacute or chronic. The more acute the disease and the more severe, the greater will be the immaturity of the involved cells in the blood stream.

MATERIALS

The present investigation includes the study of case histories of 149 cases of leukemia at the Research and Educational Hospital of the University of Illinois. These 149 cases show the following:

1. *Hyperplasia* of the gingiva was more prominent in the acute phases with the monocytic type showing the greatest incidence.

2. *Spontaneous hemorrhage* was noted in approximately one-third to almost one-half of the acute forms.

3. *Ulceration* was greater in the acute cases, the monocytic type showing the greatest tendency.

4. *Pallor* was prominent in approximately one-half of the cases with the chronic lymphatic type showing greatest incidence.

5. *Petechiae* were demonstrated in approximately one-third of the acute cases.

Most of the findings were recorded by the attending physicians over a period of approximately 15 years and many were not checked by a dentist. The tabulations were taken directly from the case histories. It is evident that the oral symptoms were more serious in the acute than in the chronic cases. The monocytic type of leukemia was the most prominent.

Biopsy specimens were obtained from 13 cases.

HISTOLOGIC AND CLINICAL FINDINGS

A biopsy specimen was obtained from a patient with a diagnosis of acute myelogenous leukemia. This patient (female) was 59 years of age. There were 80,000 white blood cells of the myelocyte and myeloblast type in her differential blood count (normal white cell count about 8,000).

The color of the gingiva was magenta.

The gingival margins were swollen.

The interdental papillae were blunted and swollen.

The surface texture was glossy and the stippling had disappeared.

The consistency was turgid.

A hemorrhagic exudate appeared upon pressure, forming dry scabs on the surface. Pockets were shallow.

A gingival papilla was removed, fixed and prepared for microscopic examination. The surface of the epithelium showed signs of dehydration and shedding. The dehydration might be explained by the fact that the patient kept her mouth open constantly—most probably because of a dyspnea (fighting for air). The prickle cell layer showed hydropic degeneration and the basal cells exhibited a tendency to proliferate evidenced by the presence of numerous mitotic figures.

The papillary and reticular layer of the connective tissue was edematous and showed dense accumulations of myeloid elements. No mature leukocytes could be seen in these sections. All cells were myelocytes and stab forms. Mitotic figures were abundant, representing most probably dividing myelocytes. Some leukocytic elements were seen migrating through capillary walls; the tissue surrounding the vessel was also surrounded by immature myeloid elements.

The blood picture as well as the sternal puncture revealed immature myeloid elements in the bone marrow and blood stream; similar findings were revealed in the gingival infiltration in the microscopic examination.

Cellular infiltrations of immature myeloid elements were found in other cases of acute myeloid leukemia.

In a case of subacute myeloid leukemia immature leukocytes were found in the exudate of one of the pockets. This patient was 57 years of age. Upon admission, the white blood count was 18,100, with 37 per cent of the cells representing the myeloblast type. The blood count rose steadily and was 130,000 at the time of the oral examination.

The color of the gingiva was red.

The gingival margins were swollen.

The interdental papillae were blunted and swollen.

The surface texture was glossy.

The consistency was boggy.

A hemorrhagic exudate appeared upon the slightest pressure.

The pockets were deep.

Employing a fine capillary tube, a portion of the fluid content of one of the pockets was procured; a drop was spread over a slide and stained by the Giemsa method. The smear showed a group of granulocytes, the majority of which were immature and probably myelocytes. The background consisted of a mixed bacterial flora, representing predominantly fusiform bacilli and various types of cocci.

In chronic cases of myeloid and lymphatic leukemia, the gingival biopsy revealed only very few immature elements in the tissues and there were no characteristic tissue changes present.

A case was observed in which the white cell count was 352,000 and the differential count demonstrated that 94 per cent of the cells were lymphocytes.

The color of the gingiva was red but the tips of a number of interdental papillae showed pallor.

The marginal contour was swollen and rolled.

The papillary contour was blunted.

The texture was glossy, stippling has disappeared.

The consistency was turgid.

There was no exudate.

Pockets were extremely shallow.

The blood smear of this case demonstrated lymphocytic elements of various stages of maturity.

The biopsy specimen revealed a chronic lymphocytic and plasma cell accumulation. It is almost impossible to make a diagnosis of lymphatic leukemia from this biopsy specimen alone, since the pathologic changes cannot be distinguished from an ordinary chronic inflammatory process. It would be necessary to distinguish the less mature forms of the lymphocytes from the adult types.

CONCLUSIONS

In conclusion we can state that the chronic forms of myelogenous and lymphatic leukemia do not produce characteristic gingival changes. The acute myelogenous type shows characteristic changes. These changes consist of a dense accumulation of immature white blood cell elements in the gingival connective tissue. A number of these immature leukocytes exhibit mitotic activity.

Some of the other tissue changes observed in the epithelium, connective tissue, and blood vessels, seem to be co-incidental and not a direct result of the systemic condition.*

General Conclusions

By BALINT ORBAN

FROM these investigations it is quite obvious that systemic diseases have their effect upon gingival and periodontal tissues. The effect varies according to the systemic disease. The tissue changes in pregnancy are quite different from those in diabetes and neither bear any resemblance to those changes associated with leukemia. An acute type of inflammatory reaction with proliferative tendencies is characteristic of gingivitis in pregnancy. A chronic degenerative type of reaction is evident in diabetes. In leukemia, especially of the acute myeloid type, the infiltration of the gingiva by immature myeloid elements was the characteristic feature. However, although the tissue changes are characteristic, with the exception of leukemia, they are not *specific* in the sense that a diagnosis of the systemic disease could be made from a biopsy. The

*[George Anday, M.D., of Chicago, well-known for his work in blood diseases was of great assistance.]

tissue changes in pregnancy, for instance, are significant only in a statistical sense; that is, the acute changes with proliferative tendencies are much more frequently found in the gingivitis during pregnancy than in any other material. The degenerative changes associated with a chronic inflammation were present in more cases of diabetic patients than in those of any other group. In a biopsy of an individual case, however, the presence of proliferative acute inflammation is merely indicative, not diagnostic of pregnancy, and that of degenerative chronic inflammation is merely indicative, not diagnostic of diabetes.

The percentage of pregnant women showing gingival inflammation was not higher than that of non-pregnant women or any other individuals, which indicates that pregnancy is not an etiologic factor in gingival inflammation; it is not even a predisposing factor, but only a modifying factor. Unfortunately, our diabetic material was not large enough for statistical evaluation, but what conclusions can be drawn, have been presented in this Symposium. As was pointed out, there was considerable loss of teeth in diabetics and there was a definite correlation between the severity of the diabetic status and the resorption of alveolar bone.

The nature of the association of diabetes and alveolar bone resorption could not be definitely established. The tissue changes in the gingiva were characterized by chronicity of the process, lymphocyte and plasma cell infiltration, in some cases hyalinization of vessels with thrombosis and degeneration of collagen fibers. Statistically, we can state that the thirty diabetic biopsies differed greatly from those of pregnant women, but one single section would permit only a guess as to the diagnosis.

The tissue changes found in the biopsy specimens of diabetics, especially the vascular hyalinization and collagen fiber degeneration, could well explain the breakdown of the supporting tissues of the teeth. This becomes especially significant if we take into consideration the ever present mechanical strain to which the supporting tissues of the teeth are subjected. Similar degenerative tissue changes were found to be the characteristic pathologic feature in the periodontal disease known as periodontosis, supposedly caused by some systemic disease.

On the other hand, some systemic disorders, such as leukemia, will produce characteristic tissue changes. An acute myeloid leukemia could be diagnosed by the presence of immature myeloid elements not only in a biopsy specimen but also in a gingival pocket smear, as Dr. Wentz has shown. Other systemic diseases, such as tuberculosis for example, also will produce specific changes. In all these diseases, however, characteristic pathologic changes will occur, not only in the periodontal tissues but also in other organs, and we cannot simply compare these periodontal symptoms of a systemic disease with other periodontal diseases. In leukemia, we should speak of a myeloid gingival infiltration. This is the gingival manifestation of a systemic disease. The same might be said of a tubercular inflammation of the periodontium. In these instances the oral manifestations are specific and are only a detail of the whole systemic picture.

In summarizing, we can state that systemic diseases can change the character of an existing gingival or periodontal disturbance so that the clinical as well as pathologic picture might be altered and aggravated. In these cases, we can speak of the systemic disease as a modifying factor. Some diseases, such as acute myeloid leukemia, regularly or frequently, will show oral manifestations and others, such as tuberculosis, syphilis, etc. will do so occasionally. In these cases, we can speak of oral manifestations of systemic diseases. In the case of diabetes, however, the influence of the disease on the supporting tissues might be more direct. It is at least probable that diabetes causes directly a degeneration of collagenous fibers and thus acts as a modifying as well as an etiologic factor in periodontal diseases. Other systemic factors such as severe vitamin deficiencies, might act in a similar manner.

The study of three systemic disorders shows, in statistical analysis, three different types of influence on the supporting tissues of the tooth.

Pregnancy modifies the local tissue reaction.

Diabetes, in part, modifies the tissue reaction; in part, however, it causes their breakdown through degenerative tissue changes.

Leukemia causes specific changes (infiltration) of the gingiva.

Home Care of the Mouth

I. Proper Home Care of the Mouth

By W. J. CHARTERS, D.D.S., *Des Moines, Iowa*

IN EVERY vital health movement today, the real slogan is "prevention". Without a doubt, "an ounce of prevention is worth a pound of cure", and nowhere else in the human body does that truth apply more forcibly than in the care of the teeth. How can we dentists feel that we have done our full duty in preventive dentistry if we consider the large number of adults who have regularly made semi-annual visits to their dentists, who have brushed their teeth the prescribed number of times each day, and who have suddenly been informed that they have well-developed cases of periodontal disease and infected teeth? In view of this condition, it is hardly surprising that some observing critics find fault with the whole dental procedure and decry all attempts to teach patients how to keep gum tissues and teeth healthy. Whose is the fault? Can something be done to insure a correct preventive technic?

In my own practice, in the examination of fifty new patients' mouths, I found that disease was not due to the disuse of the toothbrush, but to its misuse. If we dentists devoted 50 per cent of the time now spent in selling restorative dentistry to the teaching of a system of home care for the gums and teeth, we could raise our professional standing in the mind of the laity. Such teaching would do more to insure the general good health of our patients and to give us the personal satisfaction of seeing pink resistive gum tissue and spotless teeth, which condition we have helped to procure through our advice. The blame for the astounding prevalence of infected teeth and periodontal disease, among the adults who have been regular dental patients year after year, must be placed squarely on the dentists themselves.

Particularly do I think that all dentists owe a responsibility to the children in this matter of prevention. In many of our schools today, the teacher takes cognizance of the importance of care of the teeth when she gives credit for tooth-brushing among the "health chores". In many schools too, the school nurse examines the children's teeth and recommends visits to the dentist when she observes marked tooth defects. Can not the dental profession take advantage of this school interest by teaching the correct method of tooth-brushing and gingival stimulation? The correct method is difficult for the beginner and can not be accomplished without personal demonstrations, but it can be taught successfully. There is no use merely to insist in the schools on perfunctory brushing, when the results of such brushing will be nil in after life. Let us try to save the children from finding themselves in a generation, possibly 80 per cent afflicted with periodontal disease. Why should we not place on the curriculum of our schools instruction in the correct method of manipulating the toothbrush, emphasizing the health value of proper care of the teeth, at least to the same degree that we stress the importance of physical education work in gymnasiums and on playgrounds? Every dentist who feels a responsibility for the health of his community should feel a challenge in this need for dental health education work in the schools.

CORRECT PREVENTIVE TECHNIC

In scientifically teaching the use of the toothbrush for the last thirty-five years, I have proved to myself, and to many others, that to teach a patient how to wield a toothbrush correctly, so as to stimulate the gum margin all around each tooth, especially in the inter-dental spaces, is a hard and a tedious task. You will readily understand that in doing this, you cannot possibly avoid causing friction on two surfaces of the teeth, with the exception of extremely concave approximal surfaces and a small portion of the surface lying immediately below the contact area. However, the harassing treatment that the acid-forming bacteria receive at these places is unwelcome and will go far to prevent the effects of these treacherous tooth-destroying agents.

My experience in experimenting with dental floss over a long period of time has shown that it is impossible to produce resistive gum tissue by its use. It is not my intention to discourage the use of floss. I

only wish to convey to you what it will not do in my hands. (I wish to make the statement at this time that every suggestion given herein has been tried and proved by the writer.)

Next to the toothbrush in usefulness is the instrument for the stimulation of the gum margin. This instrument is made of wood. The size and shape of this instrument depends upon the interproximal conditions. Almost all individual mouths will require one or more different sizes. The size is determined by the condition of the alveolar process and the soft tissue. The instrument is made to enter far enough between the teeth that the points may be felt with the tongue, and, in this way, pressure is created on the gum margin with the aid of the contact where gum recession is not too pronounced.

Before I proceed with the explanation of my method of teaching the correct use of the toothbrush, may I say that a pathologist who worked with me in a series of tests found that the bacteria taken from scrapings between the teeth of patients under instruction showed, after three weeks of correct brushing and stimulation, to have had a reduction of from seven billion to two million bacteria.

TEACHING A NEW PATIENT

At the first appointment, have the patient bring two toothbrushes. One is for home use and the other is labeled and left in the office for instruction purposes. These lessons, which are given by a nurse, consume about fifteen minutes; ten minutes for personal brushing, using the brush to stimulate the gum margin around all of the teeth, and five minutes for demonstration on models of teeth as to how, why, and where to place the brush.

On the second visit, the nurse does the stimulating but eliminates the work on models. Instead, she has the patient take the brush, and proceeds to instruct as to how to place and use the bristles only on the outside surface of one quarter of the teeth. To cover more surface than this in one lesson would be confusing and the instructions would probably be forgotten. On each consecutive visit, another quarter section of the tooth surface area is taken, until the entire surface both labial and lingual, has been taken care of. Appointments are made, weekly or oftener, if possible, and are continued until the patient is able to acquire results without further instruction. The mouth should be rinsed thoroughly after the brushing as the brush carries out but little mucin. The use of the disclosing solution is very important as it reveals to the patient the surfaces he is missing.

A great deal has been said about the toothbrush carrying infection. I will admit that there would be cause for alarm if two people used the same brush, but I am assuming that this is not the case; so I would suggest that you transfer most of the time used in warning or debating that the condition of the brush may be harmful to instructing the patient how to apply the brush correctly. Thus, more will be gained for both the dentist and the patient.

The thorough drying of the brush after each brushing will also add to its cleanliness by discouraging the growth of any bacteria that escaped the flushing, because of their change of environment.

THE METHOD

Place the brush at right angles to the long axis of the teeth, then gently force the bristles between the teeth, being careful not to pierce the gums; that is, do not allow the points of the bristles to rest on the gums. With the bristles between the teeth, exert as little pressure as possible giving the brush several slight rotary movements, causing the sides of the bristles to come in contact with the gum margin, producing an ideal massage. Be careful not to make this movement sufficient to remove the bristles from between the teeth. After making three or four small circles, remove and replace in the same area. Make three or four applications in the same place. The reason for this is that the points of the bristles strike the labial, buccal or lingual surfaces, then slide into the interproximal spaces. Move the distance of one embrasure, and repeat the process, holding the sides of the bristles firmly on the gum margin. The V shaped space between the teeth assists in crowding the sides of the bristles against the gingivae. Where teeth are missing, we must depend on the sense of touch to keep the proper pressure, with the sides of the bristles on the gum margin.

II. Home Care Technics—Ancient and Modern

By DOROTHEA F. RADUSCH, D.D.S., B.A., M.S., *Minneapolis, Minnesota*

THE PROGRAM CHAIRMAN suggested that I introduce my discussion on home care by a brief historical review. Historical records indicate that many centuries ago, before the Christian Era, oral hygiene was practiced to a marked degree. No doubt food impactions between the teeth produced discomfort for these early peoples, just as occurs today. For relief, they improvised crude toothpicks of wood or quills. Much later, elaborate and ornamental toothpicks were popular.

The early teachings of oral hygiene were a part of religious training and ritual. The Buddhists in the Fifth Century B.C. used a "tooth stick", a kind of brush made from a fresh branch. Likewise the Mohammedans used, in obedience to religious prescription, the "miswak". (1) This is made as follows: "From the end of a properly cut little stick of wood, flexible and knot-free, a piece of bark is removed. After the stick has been soaked in water for twenty-four hours, the peeled portion is pounded with a stone or hammer until the plant fibres unravel to form a kind of small paint brush. The unbeaten part of the stick serves as a handle." "You shall clean your mouth for that is the way to praise God," Mohammed said. (2) He gave detailed rules for the proper grasp of the miswak, and procedure in cleaning. The labial and buccal surfaces were cleaned first, and then the "brush" was guided into the interdental spaces.

It is reported that Pliny who lived in 23-79 A.D. said, "It makes teeth firm to pick them with a porcupine quill" and again, "Use the bone of a hare to prevent bad odors of the breath." (2)

The Hindus have many rules for health and beauty in their book about love and marriage, *Kama Sutra* which was written in the First Century A.D. They recommended in this book the chewing of a certain kind of wood to clean the teeth in order to improve the relation between husband and wife. The eating of betel leaves is recommended to give fragrance to the mouth.

All through medieval times in Europe, coarse cloths or napkins were in use for cleaning the teeth, many times along with aromatic dentifrices, including sage leaves. Recommendations were made too for rubbing the teeth with the fingers.

Still unknown to Western Civilization, the first tooth brush with real bristles standing at right angles to the handle, had been constructed in China about 1500. The inventor is unknown. The first mention of a toothbrush in Europe is recorded in a German dictionary published in 1719, while one of the earliest pictures on record is dated 1818. Even that long ago, users were cautioned against careless use of these brushes, which would cause undue wear of tooth structures.

Hirschfeld, in his book on *The Toothbrush, Its Use and Abuse* includes a detailed historical review. He relates that Gariot in 1843 discussed the merits of hard and soft brushes. Quoting Gariot "Delicate females, who take great care of their mouths, and whose teeth are easily cleaned, should use a soft brush. Men, who clean their teeth but seldom, require a hard one. It will be found convenient to have an assortment of brushes, so as to use such as may be most proper under different circumstances."

We, in these modern times, have added comparatively little to the fundamental knowledge of oral hygiene. Our clinical experience has demonstrated that subgingival cleansing of teeth has limited value unless supplemented by properly directed brushing. Thus brushes should be on hand in the office along with all other instrumentarium for use in treatment. We believe that a good brushing technic is an aid in healing, and furthermore, that it is a significant factor in prevention of much gingival disease, and in maintenance of healthy tissue. The patient is the one who must apply the daily care, and he must be willing to give the necessary time each day if favorable results are to be accomplished.

At first, when the patient is worried about the possibility of losing teeth, there is often enough motivation to bring about strict attention to the dentist's instruction. Even so, the method should not be too long and tedious, or the patient will become discouraged. The mouth-care habits established at this time must be followed continuously so long as natural teeth are present.

Inquiry into habits of brushing when treatment is begun reveals that methods in common use are usually haphazard, super-

ficial, ineffective, or even harmful. Thus most patients require detailed instruction. The brushing technic I teach is not original nor unique, but one described in many books and articles. It is very similar to that of Dr. Charters. I teach interdental brushing, starting on the lower anterior labial, since here the patient can see most easily the placement of the brush at about a 45 degree angle with bristles sloped away from the gums. I ask that the brush be moved slowly in a circular or slightly up and down motion to a count of fifteen, cautioning against such heavy pressure or speed that movement of the bristles is arrested. Also, I do not have the patient remove the brush from position, once it is properly placed, until it is placed for a new group of teeth. The right-handed patient proceeds to the left, continuing around the last tooth present in the arch to the lingual, shifting at this time to application of the toe of the brush instead of the entire brush. In brushing the lower anterior lingual tooth surfaces, I ask the patient to keep the brush at an angle, which permits better access in the vulnerable areas. At the right cuspid region, the brush is pivoted to duplicate the left placements. After completing the entire lower jaw, the brush is transferred to the upper, reversing the brush in the same relative positions. Individual special placements may be necessary to reach teeth showing marginal horizontal variations. After brushing, vigorous rinsing to eliminate dislodged material and dentifrice is advised. Some patients are quick to master the system, others require repeated encouragement. Not all become equally efficient.

Only occasionally do I suggest any other than interdental brushing for adults. In selected instances I may teach occlusal brushing or massage of the gums by brush. If a satisfactory interdental technic is mastered, I find the other steps superfluous, unless a tooth does not have a functional opponent, or there is excessive fibrous hypertrophy.

When gingival tissues are highly edematous, or inflammatory, I may use a hard or medium unbleached bristle brush at first. In all other instances I recommend the hard black natural bristle, which if properly placed and vibrated does not injure the tissues, and is much more effective for the time and energy expended.

Whenever there has been sufficient interdental loss so that there are open areas interdentally, I recommend regular use of a right angle stimulator.

On the basis of reasoning, I am convinced that a dentifrice is an aid in producing a clean mouth, just as soap is an aid in cleansing the hands, but proper application of friction, sufficient but not abrasive, is required in each instance.

The patient is more likely to be impressed by the need for excellent home coöperation if the dentist takes the time to teach this phase, instead of delegating it entirely to auxiliary personnel. Since most patients require a number of lessons before learning a satisfactory technic, instruction is given both by the assistant and myself. The patient is not released from rehearsal of brushing until I have accepted his technic. In some instances, disclosing solution for home use is an excellent method of helping the inept patient visualize his inadequacies. It is always used at the office to show the patient how poorly or how well he is brushing. At all times we aim for maximum benefit in cleansing and stimulating actions of the brush, with a minimum of traumatization.

It is not sufficient to depend upon the instruction given in the first series of visits. For continuous excellence in home care, the patient's technic should be observed at subsequent periodic recall appointments. More particularly, this should be done whenever there is evidence of dental calculus or marginal hemorrhage. Without repeated instruction, some patients find it difficult to maintain a satisfactory system.

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III. Teaching Home Care to the Patient

By DICKSON G. BELL, D.D.S., *San Francisco, Calif.*

MENKIN (1) points out that inflammation is a manifestation of a cellular injury brought about by the presence of an irritant. All local periodontal disturbances have their origin in inflammation and several of the most common irritants are calculus, materia alba and food impactions. An inter-relationship exists between these factors and the prevention or control of them is in the hands of the patient.

Gardner (2) states, "In a questionnaire submitted to 1000 patients, less than 10 per cent had received instruction in mouth care from a dentist and fully half of these were instructed in ineffectual methods". Clinical observation would certainly support this statement. Therefore, it is necessary to teach correct mouth hygiene to all of our periodontal patients. The most effective method of instruction is to take the toothbrush, dental floss or interdental stimulator of some type and actually demonstrate their use in the patient's mouth.

All of us will agree, I am sure, that the best known agent for cleaning the exposed surfaces of the teeth is the bristle brush. There is, however, no general agreement among dentists regarding the toothbrush as indicated by the many different shapes and sizes, and degrees of bristle texture that are being manufactured today. Unfortunately, this situation causes the public to assume that it does not make much difference what toothbrush they use. Some patients have even been known to select a toothbrush because the color of the handle matched the color scheme of their bathroom.

The selection of a toothbrush should be made on the basis of an analysis of what one wishes to accomplish, namely, the removal of the materia alba and food debris adhering to the surfaces of the teeth and the development and maintenance of good gingival tissue tone. The shape of the dental arch, the position, contour and size of the individual teeth are such that the bristle surface of the brush cannot be trimmed to fit the teeth. A brush that would fit the molar teeth would not be suitable in the anterior region and one that was shaped for the labial convexity in the cuspid area would certainly not fit the concavity of the arch on the lingual. In most instances, it is not possible to thoroughly brush more than two or three teeth at one time, and with the exception of the cuspid, such groups of teeth will generally conform to a straight line rather than a curved one.

In view of these facts, it is this author's opinion that the most satisfactory type of brush is one that has a flat or level trimmed bristle surface—the so-called "straight-toothbrush". It should be of medium size with a large number of bristle knots or tufts set close together and four rows in width. This gives a degree of density or body to the brush. The many bristles will cover more area of the tooth surface and thus give a better cleansing result. The bristle surface of this brush should be 1½ inches in length and ⅜ of an inch in width. Since the trend of the profession is toward small brushes, this may seem too large, but the observation of many years of practice has shown that the average patient has no difficulty after he has used it for a few days. There may be a few areas in the mouth where the bristles at the heel of the brush have no cleansing action but they often act as a "brake" where there is a tendency to be too vigorous with the brushing. However, a smaller size brush 1¼ inches in length is recommended for patients with a small mouth and for children. The bristles should be of fine texture and preferably those obtained from the wild boar since up to the present time no satisfactory brushes with nylon filaments have been manufactured. The length of the bristles should not be less than 7/16 of an inch or greater than ½ inch, depending on the type of filament to provide the correct amount of flexibility. These are important factors where the brushing procedure is chiefly in the vertical direction since short bristles or those of large diameter will not properly clean the surfaces of the teeth, but frequently traumatize the gingival tissues.

The brushing procedure is that which was recommended some years ago by T. Sydney Smith (3). Patients are taught to always brush their teeth in a routine manner, beginning with the buccal surface of the last tooth on the lower left side of the arch. The dentifrice is applied to the

brush, which has been previously moistened with tepid water, and placed against the buccal surfaces of the last teeth. With the mouth slightly closed to relax the cheek muscles, the handle of the brush is held in a horizontal position with the bristles at right angles to the teeth and using a gentle medium stroke, the bristles are directed down over the buccal surfaces of the molars. It may also be described as a sort of sweeping motion. After a series of strokes, the brush is moved forward to the bicuspid region and the same procedure is followed, then the cuspid-lateral area, and this is continued to the last tooth on the opposite side. When it is done correctly, about six to eight brushing strokes are generally required to thoroughly clean each group of teeth.

On the lingual surfaces of the lower molars and bicuspids, it is difficult to use this sweeping motion because of the proximity of the tongue and tissues which form the floor of the mouth. Therefore, with the handle of the brush held at a slight angle above the horizontal position, these teeth are brushed first with a downward motion and then followed by several horizontal strokes to make certain that the tooth surfaces at the gingival margin have been thoroughly cleansed. For the lingual surfaces of the lower anterior teeth, the brush is held in a vertical position and the direction of the bristle strokes is both upward and downward. The procedure of brushing the right posterior teeth is the same as that used on the left side. Heavy deposits of calculus are usually found on the lingual surfaces of the lower anterior teeth and a brush of this width will remove the first deposits of soft material better than the average type of toothbrush. Since the brushing in this area is in a vertical direction, the extra number of bristles permits some to conform to the lingual contour of the incisor teeth, while others enter the interdental spaces and produce a stimulating reaction in the gingival tissues as they pass down over them.

After the brushing of the lower teeth is completed, the patient is instructed to clean the brush in water and apply a fresh amount of dentifrice for the upper teeth. Probably the most neglected areas are the distal and buccal surfaces of the upper second and third molars. Observation of most patients' brushing technic has shown that they invariably open their mouths as wide as possible and start the brush in motion as they introduce it into the mouth. The result is that they usually begin brushing about the first molar region. This problem may be corrected by first having the patient relax the cheek muscles and then place the brush in position against the buccal surfaces of the molar teeth. Commencing again on the left side and with the brush in a horizontal position, the disto-buccal surface of the last molar is cleaned with a short circular motion, together with a few horizontal strokes of the brush. On the buccal and labial surfaces of the upper teeth, the direction of the bristles is with an upward motion. However, some patients find it difficult to use the brush effectively in this manner and they are advised to cleanse these surfaces with an upward and downward stroke. The procedure of only brushing a few teeth at one time is followed, beginning with the molars, then the bicuspids and continuing around the arch to the molars on the opposite side. The disto-buccal area of the last right molar is cleaned in the same manner as was done on the left side. The method of brushing the lingual surfaces of the upper teeth is the same as for the lower, except that the handle of the brush is held slightly below the horizontal position for the posterior teeth. Finally, the occlusal and incisal surfaces of all teeth are thoroughly brushed with a circular and horizontal motion to remove any food debris. The patient is instructed to test all surfaces of the teeth with the tongue, if any of them do not feel smooth, some dentifrice should be applied to the brush and these particular teeth cleaned again.

It is the author's opinion that the same brushing technic cannot be effectively applied to all surfaces of the teeth. It is necessary for the patient to change the position of the brush and the direction of the brushing strokes for the different sections of the arch. Excessive pressure should be avoided, and rapid bristle strokes are inefficient. The brushing should be done with a moderate degree of pressure which the tissues will easily tolerate without injury. The maintenance of good tissue tone is accomplished since most of the brushing strokes are directed down over the lower gingival tissues and up over the upper gingiva, thus stimulating the venous flow of blood. This prevents anoxia and stasis in the gingival tissues. While the need for tissue stimulation

is recognized, yet gingival massage is not emphasized in toothbrush instruction because the movement of the brush in cleansing the gingival portion of the tooth surfaces will at the same time direct a sufficient number of bristles over the margin of the gingiva. "Gum massage" has been so widely publicized that many patients have the idea that it is the all-important factor. "I always massage my gums" is a statement that is frequently heard. This tendency to brush the gingival tissues often causes abrasions and irritation. It undoubtedly accounts for the many cases of recession of the gingivae, especially when nylon or hard natural bristles are used.

Frequent application of the dentifrice during the brushing, whether paste or powder, will accomplish better cleansing results than when a large amount is used only once or twice. In the latter instance, the teeth which are first brushed receive most of the benefit of the dentifrice and the remaining ones practically none at all. Since the brush is made of natural bristles, it is recommended that patients have two brushes which are to be used at alternate times. This gives the first one an opportunity to dry out before being used again. It has been previously stated that a stiff toothbrush is not desirable and neither is a wet, soggy brush which does not have the proper flexibility. Patients are also advised to carefully wash the brush of dentifrice and saliva, dry it on a towel, and keep it in the air or exposed to sunlight if possible. Thus they always have a clean toothbrush and not one that is discolored and the bristles packed with dentifrice.

Such a detailed description of this method of toothbrushing may lead to the assumption that it consumes a great deal of time and is an arduous task for the patient. In reality, it takes but a few minutes when done in a routine and methodical manner. Most patients have acquired many bad brushing habits and it usually takes several periods of instruction before they overcome them. There are always a few patients who do not thoroughly brush their teeth, yet they state that they have implicitly followed instructions. Apparently, they gaze about the room or are otherwise distracted. In such cases it is suggested that they close their eyes while brushing their teeth. This forces them to concentrate on what they are doing at that time. The true test of any procedure is what happens over a period of years. The correct use of this type of toothbrush has resulted in some decrease in dental caries, there is a minimum amount of irritation or recession of the gingival tissues, and almost no evidence of tooth surface abrasion. Many patients are under observation who have continuously used this method for over twenty years.

Good mouth hygiene means not only the correct use of the toothbrush but also thorough cleansing of the interproximal areas. Several different agents are used for this purpose—dental floss, dental tape, soft wood points and soft rubber cones. Each has its particular indication, depending on the height of the septal gingiva or the size of the interproximal space. When the crest of the septal tissue is near the contact point, the patient is advised to use dental floss. If there is a moderate loss of tissue, the distance between the contact and the gingival crest is greater, the space wider, and dental tape is recommended. Patient instruction in the correct use of floss or tape is very important, since a lack of control in passing it through tight contacts will result in traumatizing the septal tissues. The patient should hold the floss or tape firmly between the thumb and index finger of both hands, carefully work it between the teeth, relaxing the finger pressure as soon as it is felt passing through the contact and with a bucco-lingual motion, cleanse the proximal surfaces of the teeth from the crest of the septal tissue to the contact point. Soft wood points have been recommended in some cases where the contacts are not ideal but broad and unusually tight so that the patient could not manipulate the floss, tape or tip of the rubber cone. The soft wood being of a fibrous nature cleanses these surfaces in an excellent manner, but care must be taken not to wedge it between the teeth or irritate the soft tissue. One objection to these soft wood points is that the point will buckle rather easily if not carefully inserted between the teeth. In patients having extensive loss of the septal tissue or where gingival resection was necessary, the use of a rubber cone is recommended. The soft rubber cone will cleanse the large interproximal space and proximal surfaces of the teeth more thoroughly than either dental floss or tape. It also has a stimulating effect on the septal

tissue when used with a gentle up and down motion. There are some patients who have no manual dexterity, they are clumsy with their fingers and have difficulty using dental floss or tape, but are able to manipulate this instrument with satisfactory results. All patients are advised to do their interproximal cleaning in front of a mirror. They are more likely to use the different agents correctly, follow a routine procedure and not miss any areas since they are able to see what they are doing.

The amount of time a patient should spend on home care will of course depend on several factors, such as the number of teeth present, malposition of teeth, and prosthetic appliances. The average mouth will require about four minutes for thorough brushing and interproximal care. From an ideal standpoint, the number of times a day that this should be done would be on arising, after each meal and before retiring. However, experience has shown that many periodontal patients find this procedure somewhat impractical because of occupation, available time, and convenience. Therefore, they are advised to brush their teeth and use dental floss or other interproximal cleanser at least twice a day—night and morning, and the most important time in the morning is after breakfast. These are the two periods when most patients have sufficient time to do it properly. Those who are not able to brush their teeth after each meal are advised to do whatever is possible to maintain a clean mouth, such as vigorous flushing with water to remove loose particles of food; this is especially important in those cases with removable appliances. There are many variable factors in the daily lives or routine of patients and the instructions for the care of the mouth during the day must be considered from this standpoint. The principles of correct oral hygiene are discussed with the patient during periodontal treatment but the actual demonstration in the mouth and the patient's application of them are deferred until the inflammation has been reduced and tissue repair has taken place. The patient is not dismissed until he has demonstrated his ability to properly care for his teeth and investing tissues.

The cleansing of the tongue should be recommended as a daily procedure in mouth hygiene. Patients are advised to examine the dorsum of their tongues and remove any soft accumulation. The level trim toothbrush is an excellent agent for this purpose, eliminates the need for having one more prophylactic agent, and it takes but a few seconds to gently clean the surface. Other designs of toothbrushes and especially those with stiff bristles are not suggested for this procedure as they are likely to cause injury to the mucous membrane of the tongue.

The objective of periodontal therapy is to reduce inflammation, bring about a repair of the lesion, and reëstablish function. The objective of home care is to maintain this condition as long as possible and the degree of success that is obtained will depend on the coöperation of the patient. Since he has been taught a routine method which embodies certain desirable habit-forming principles of oral hygiene, it then becomes his responsibility to carry out these measures. The significance of this fact must be fully emphasized to the patient. Correct mouth hygiene is a therapeutic procedure, equal if not greater in importance than that of treatment of the periodontal lesion.

Booker T. Washington in his book *Up from Slavery* states "In all my teaching I have watched carefully the influence of the toothbrush, and I am convinced that there are few single agencies of civilization that are more far reaching".

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Editorials

The Chicago A. A. P. Meeting

IN THE current issue of the *Journal of the American Dental Association* (p. 477) the following notice appears:

Periodontists.—The American Academy of Periodontology, meeting for the thirty-second time, elected Dr. Edward L. Ball, of Cincinnati, president to succeed Dr. Raymond E. Johnson, of St. Paul. Other officers elected are: Drs. Dickson G. Bell, of San Francisco, vice-president [president-elect]; Clarke E. Chamberlain, of Peoria, Ill., secretary and Dr. C. W. Marriott, Cleveland, O., treasurer.

To this notice of our regular three-day annual meeting, one might add that hundreds of dentists from all sections of the country, assembled to further the study and understanding of the various diseases and influences that tend to destroy the periodontal tissues. The program was so planned that it was of interest to the researcher, the teacher, and the clinician (dentist and periodontist). The audience gave serious attention to both the scientific and practical subjects. Two of the Symposiums presented are published in this issue of the Journal. Other papers read will appear in later issues. The arrangements were taken care of in an efficient manner, even to the last detail, which is a real accomplishment in these days of road blocks in whatever direction one is going.

Periodontia is of such basic value to all dentists interested in the preservation of the natural teeth, that the Academy meetings should be attended by thousands instead of hundreds, since they are open to all members of the A.D.A.

While no dental meeting is ever 100 per cent satisfactory to all who attend, the 1948 meeting came very near to surpassing all former standards.

It has not been customary for the Journal to publish the annual Academy Presidential Address because of its usual specific relation to the interests of members only. Dr. Raymond E. Johnson's address, however, stressed two subjects of general interest which are appropriate to mention here. First, is the close inseparable relation of periodontia and preventive dentistry and consequently the relation of periodontia to public health. Periodontists who have stressed the importance of home care and follow-up appointments, and have observed cases over periods of thirty to forty years, have been impressed with the small number of restorations needed by these patients. Oral hygiene is incomplete which does not include the gingival sulcus (the dentist's responsibility), the interproximal spaces, the proximal and all other tooth surfaces, the care of the tongue and the encouragement of normal circulation in the gingiva by any harmless means. For the preservation of the natural teeth, the periodontist knows that periodontal health is an essential part of *preventive dentistry*.

The second subject emphasized by Dr. Johnson was the need for a "generally accepted classification of periodontal diseases". We have long since passed the time when these diseases were divided into two classes, viz., Vincent's disease and "pyorrhea". This Journal has previously published suggested classifications. At the Round Table Luncheon, Dr. Maynard K. Hine presented a classification in printed form and asked members and guests to make suggestions in writing on them. These were collected for study. Many teachers of periodontia were present and it is hoped that a classification which will be generally accepted will evolve from this effort. A correct classification is bound to lead eventually to proper treatment for the various conditions.

To contribute to the proper classification, a clear understanding of terms used in periodontia is essential. The Report of the 1948 Nomenclature Committee is published (*See* p. 147), although it is quite similar to the report published in 1947. It is important to keep these terms before our readers in the hope that they will be commonly used.

Ties of Sacred Honor

THE REPORT of the Committee on Ethics read at the September meeting left many Academy members aghast. Not that the Report contained new information, but an unethical practice had been revealed through a reliable source, and officially stated in this committee report. Periodontia was being exploited!

Surgery has been glamorized over the years and associated with it the idea of fees of sizable proportion. In medicine, human life is often at stake and it is quite understandable that no surgeon cares to assume that responsibility without adequate compensation. Patients are easily impressed by surgical procedures but to the layman subgingival curettage resembles the "tooth cleaning" process, which even many dentists despise and hesitate to meet its exacting demands in technical training.

If the treatment of periodontal diseases consisted solely of eliminating periodontal pockets, surgical removal should be even *less* costly for the patient. It is feared that many dentists have this incomplete idea of periodontal treatment and are practicing it accordingly, if one is to judge by the many complaints received by the Committee on Ethics. The simplicity of the operation, and the chances for a sizable fee have no doubt influenced many who have had no other preparation for the practice of periodontia and who have never been previously interested in it. Regardless of the explanation—"He should refrain from the discussion of his fees with his colleagues, and from boasting of their size. To do so is unprofessional and in bad taste." *From* Code of Ethics, Section 111—American Academy of Periodontology.

Precocious Advanced Alveolar Atrophy

By SAMUEL CHARLES MILLER, D.D.S., F.A.C.D.*

PERIODONTAL disease may, and often does, have its inception in early childhood. As methods of diagnosis improve and the incipient signs of periodontal disease become generally known, more and more cases in youngsters will be reported. A study by Miller, Wolf and Seidler (1941) indicates a specific alveoloclasia of youth, which they named precocious advanced alveolar atrophy. The condition was usually found associated with an elevated serum cholesterol and serum calcium, and a flattened sugar tolerance curve. The patients that were studied also favored a heavy carbohydrate diet over a long period of time either as a cause or a result of the low blood sugar.

The advanced alveolar loss revealed roentgenographically in early youth may create a pessimistic view and invite wholesale extraction. Yet, upon careful analysis, it will be found that even though some teeth are badly involved, others may at the time of examination have as yet escaped serious alteration. Three classes may be differentiated:

Class	*Teeth Involved*	*Period of Activity of Causal Agent*
1	6 21 | 12 6 6 21 | 12 6	5 to 7 years of age
2	7 543 | 345 7 7 543 | 345 7	9 to 12 years of age
Combined	All	Continuous

In the first type which is most common, the cause may be endocrine disturbance, inherent bone weakness, malnutrition or extreme emotional upset causing clenching at the age when formal schooling starts. The causal agent had been in operation at the time when the alveolar support of the incisors and first molars was being formed. Those teeth which erupt later are well supported by bone. The incisors, even though they cannot be ideally handled, will improve under orthodox treatment. The important decision involves the handling of the first molars. Since these develop trifurcation and bifurcation involvements early, their prognosis is often hopeless at the time the patient is seen.

It is much better to extract at an early age an upper first molar with trifurcation involvement and thus protect the second and third molars and to a lesser extent, the bicuspids. If a conservative approach is taken and the first molar is allowed to remain the bone involvement will extend to the other molars and cause their early loss (Miller and Seidler; 1942).

The construction of a stationary bridge for the first molar space, rigidly held by soldered joints to both contiguous teeth, will prevent loss of bone on these teeth and in many cases where the loss has already occurred will allow regeneration to take place.

*Professor of Periodontia and Chairman of Periodontia Department, New York University College of Dentistry, New York, N. Y.

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Nomenclature in Periodontia

(REPORT OF THE NOMENCLATURE COMMITTEE OF THE AMERICAN ACADEMY OF PERIODONTOLOGY)

*Read at the Thirty-second Annual Meeting September 9, 1948, Chicago, Illinois

THE RAPID ADVANCE of knowledge of diseases which affect the supporting structures of the teeth necessitates a standardization of the nomenclature so that dissemination of this knowledge can be brought to the dental profession at large. Vague terminology hinders such an effort. Only through accurate definitions of the terms employed can this difficulty be overcome; thus when one dentist employs the term "Periodontitis" or "Periodontosis", another will understand the disease process the first wishes to describe. Not only is it necessary to standardize the terminology for the various entities of periodontal disease, but also the technics employed to treat them.

The committee on nomenclature of the Academy of Periodontology this year has undertaken three projects: first, to review the periodontal nomenclature; second, to make a survey on the nomenclature used in the dental schools; and, third, to suggest a nomenclature for technics in the treatment of periodontal disease.

Because of the extensive work of the previous nomenclature committees, there has been considerable uniformity in the periodontal nomenclature. After consultation of the committee, the report presented in 1947 was re-accepted, except for some difference in a few instances of the definition of the accepted term.

In this report we will repeat the accepted terms as they were published in July, 1947, including certain modifications in definitions.

1. *Alveolar bone:* The alveolar processes of the maxillae and the mandible. Within the alveolar bone, a distinction should be made between the "alveolar bone proper" and the "alveolar supporting bone" due to their biologic and functional differences. The alveolar bone proper may be defined as that thin perforated bony plate surrounding the root to which the fibers of the periodontal membrane are attached and through which the vessels and nerves enter and leave the periodontal membrane. In radiography this bony plate is called "lamina dura" as it appears more radiopaque than the surrounding spongy bone. The alveolar supporting bone consists of the compact alveolar plates and of the spongy or trabecular bone of the alveolar processes, other than the alveolar bone proper.

2. *Alveolus, dental:* The socket in the alveolar process in which the root of a tooth is held by the fibers of the periodontal membrane.

3. *Bruxism:* Gnashing or grinding of the teeth in other than normal function. This grinding of the teeth is an unconscious habit and is often limited to the sleeping period, but may occur at times of mental or physical concentration or strain. Bruxism may develop into a neurotic symptom, "bruxomania".

4. *Calculus:* See—dental calculus.

5. *Dental Calculus:* (synonym—tartar). Calcareous deposit on the teeth (hard incrustation on the teeth, consisting of organic and mineral matter). Much confusion has existed due to the distinction between salivary and so-called serumal calculus, one said to originate from the saliva, the other from serum of the blood. This differentiation, however, is untenable and we can only distinguish between supra-gingival and subgingival calculus. The differences in consistency and color are due to the fact that in the pocket the deposits are more densely packed and are darkly stained from seepage of blood.

6. *Epulis:* The term "epulis", denoting a tumor on the gingiva, is a general term. The word originates from the Greek epi (on) ulon (gingiva) and means growth on the gingiva. It is a tumor of varying cell types, at times fibrous, at other times of giant cell character, vascular or inflammatory, etc. A proper differential diagnosis is only possible by biopsy.

7. *Gingiva:* That part of the oral mucous membrane that covers the alveolar processes of the jaws and surrounds the necks of the teeth.

8. *Gingival hyperplasia:* Overgrowth of the gingival tissues. There is some misun-

derstanding as to the correct use of the terms hyperplasia and hypertrophy. A sharp and definite difference does not always exist. However, hyperplasia is defined as an overgrowth of tissue due to increase in the number of its elements; this overgrowth does not serve a useful purpose. Hypertrophy, on the other hand is an increase of an organ due to increase of the *size* of its elements, serving a useful purpose. An example of a true hypertrophy is the enlargement of the heart due to overwork, or enlargement of muscles due to increased activity. Each cell becomes longer and thicker. Overgrowth of the gingiva, however, is a hyperplasia (increased number of cells).

9. *Gingival pocket:* The gingival pocket may be defined as one whose base lies coronal to the alveolar crest.

10. *Gingival recession:* Progressive exposure of the root surface by apical shift of the gingiva, following resorption of the alveolar crest and loss of the corresponding part of the periodontal membrane. (*See*—periodontal atrophy.)

11. *Gingival sulcus:* The shallow groove around a tooth, bounded on one side by the surface of the tooth and on the other by the epithelium lining the free margin of the gingiva. This term designates the "normal" condition as distinguished from the gingival and periodontal *pocket.*

12. *Gingivitis:* Inflammation of the gingiva. The condition may be further defined and described by such adjectives as acute, chronic, purulent, ulcerative, hyperplastic, scorbutic, etc.

13. *Intrabony pocket:* The intrabony pocket is one whose base is apical to the bone crest, the pocket extending between the tooth surface and the bone; this latter type may be narrow or broad in nature.

14. *Oral Mucosa* (or oral mucous membrane).

Masticatory mucosa: Epithelium hornified, immovably attached

Gingiva
Hard Palate

Lining Mucosa: Epithelium non-hornified

Loosely attached
Firmly attached

Specialized mucosa: Back of tongue (See Orban, B. and Sicher, H.: "Oral Mucosa," *J. Dent. Educ.* 10:94-103, 163-164, 1946.)

It is of special importance to differentiate between the gingiva (hornified, firmly attached) and the lining mucosa covering the alveolar process (non-hornified, loosely attached); these two different tissues are separated from each other by a festooned line.

15. *Periodontal abscess:* Acute purulent exacerbation of a chronic periodontitis, associated with deep periodontal pockets.

16. *Periodontal atrophy:* Progressive resorption of alveolar crest with loss of the corresponding part of the periodontal membrane and gingival recession.

17. *Periodontal diseases:* Collective term designating all periodontal disturbances.

18. *Periodontal membrane:* The fibrous tissue that connects the cementum of the tooth to the surrounding structures.

19. *Periodontal pocket:* A periodontal pocket may be defined as a space bordered on one side by the tooth and on the other by the crevicular epithelium of the gingiva. Its base is limited by the epithelial attachment. The chief features are the ulceration of the crevicular epithelium and the inflammation of the gingival corium. The pocket is usually filled with debris and calculus.

20. *Periodontal traumatism:* Injury of periodontium due to excessive occlusal operative, accidental or orthodontic stress. (*See* —traumatism.)

21. *Periodontia:* The branch of dentistry that deals with the science and treatment of periodontal diseases. Synonym: periodontics.

22. *Periodontist:* One who is learned in the science of, periodontology and skilled in the treatment of periodontal diseases.

23. *Periodontitis:* Inflammatory disease of the periodontium. *Periodontitis simplex* —sequela of chronic gingivitis. *Periodontitis complex* due to systemic disease or secondary to periodontosis. The condition may be further defined and described by such adjectives as acute, chronic, ulcerative, purulent (suppurative), etc.

24. *Periodontium:* The investing and supporting tissues surrounding the tooth, namely the periodontal membrane, the gingiva, and the alveolar bone.

25. *Periodontology:* The science and study of the periodontium and periodontal diseases.

26. *Periodontosis:* Non-inflammatory destruction of the periodontium originating

in one or more of the periodontal structures, characterized by migration and loosening of the teeth in the presence or absence of secondary epithelial proliferation and pocket formation or secondary gingival disease.

27. *Pocket: See*—gingival pocket, periodontal pocket, intrabony pocket.

28. *Traumatism:* Morbid condition of tissue caused by mechanical trauma (pressure necrosis and its consequences; thrombosis, hemorrhage, resorption of bone and cementum).

29. *Vincent's infection:* It is recommended that this designation of a certain type of gingival inflammation be discarded. The term was used to designate an acute ulcerative gingival inflammation. Properly we should speak of an "acute, sub-acute or chronic necrotizing ulcerative gingivitis". The term "Vincent's angina" should be discarded.

The second project of the committee was to make a survey on the nomenclature used in the dental schools. It revealed that out of thirty-four responses, twenty-one follow the terminology advocated by the Academy; three use the terminology of the Academy but have slightly different connotations to some of the terms; five use their own terminology; and five are indefinite. It is hoped that future committees will keep in contact with the dental schools informing them of official changes that are adopted by the Academy.

The third project was to formulate a glossary for the technics used in periodontal therapy; the following terms are listed:

Prophylaxis: The cleansing and polishing of the teeth for prevention of periodontal disease.

Odontexesis: The cleansing and polishing of the teeth as a method of therapy.

Sub-gingival curettage for gingival pocket.

Gingivectomy.

Gingivoplasty: the changing of the gingival architectural form.

Sub-gingival curettage for intrabony pocket.

Flap operation.

The committee also recommends that the report for the symbolic designation of teeth published in the Journal of the American Dental Association, Vol. 32, May 1, 1947 be adopted by the Academy of Periodontology as an official method for charting teeth.

It is also suggested that a copy of this report be submitted to the periodontal department of the dental schools.

Respectfully submitted,
Herman Becks
Maynard K. Hine
Isador Hirschfeld
Dorothea F. Radusch
Henry M. Goldman, Chairman

Dr. Edward L. Ball newly elected president, A.A.P. receives the symbol of the office from Dr. Raymond E. Johnson, September 11, 1941.

Nutrition and Dental Health

Articles *Selected* and *Condensed*

By DOROTHEA F. RADUSCH, D.D.S., B.A., M.S., *Minneapolis, Minnesota*

Dynamics of Protein Metabolism

Condensed from *Nutrition Reviews* 6:213, July, 1948.

E. P. BENDITT and his associates (*J. Lab. Clin. Med.* 33:257, 1948) have sought to determine the interrelation between the level of protein intake, the energy intake, and the utilization of dietary protein under controlled conditions. They studied the rate of utilization of protein for tissue fabrication by protein depleted animals under three different circumstances: (1) varying caloric intake with constant protein intake; (2) varying protein intake with constant caloric intake; (3) simultaneous variation of caloric and protein intakes.

Young adult male albino rats were fed for 2½ months on a low protein diet which contained adequate calories, vitamins, minerals and other dietary essentials. Weight averages were reduced from 210 g. to 154 g. Comparable groups were then selected for experimental diets. Consumption was measured daily for 14 days. Animals were then killed and analyses made.

At the lowest level of caloric intake (560 calories per square meter per day) with a constant protein intake of 1.56 g. per day, protein utilization was poor. With increasing caloric intake, protein utilization rose until a maximum was reached at an intake level of 1240 calories per square meter per day. Beyond this point there was no additional utilization of protein despite the caloric intake being increased up to 1840 calories per square meter per day.

Varying protein intake at a constant caloric intake (1630 calories per square meter per day) was associated with increased protein utilization as the protein intake was raised from 2.8 to 14.8 g. per kilogram per day. Beyond 6 g. per kilogram per day, the rate of protein utilization became progressively less.

When increasing quantities of the same diet were fed to the depleted animals with thus, a constant ratio of protein to energy in the diet, the rate of protein utilization increased with increasing diet intake.

There appears to be a restriction in the utilization of a constant quantity of protein when the caloric intake falls below a critical value of about 1240 calories per square meter per day. At caloric intakes below the critical level the animal must resort to burning protein for purely energy purposes. As a consequence, the restriction of caloric intake below the critical point has the same net effect as a restriction of the protein intake.

Other studies are described (E. P. Benditt *et al.*, *J. Lab. Clin. Med.* 33:269, 1948) in which protein intake ranges commonly used and feasible in human feeding were employed. The diet given these rats offered approximately 0, 1, 2, 3, 4 g. of protein per kilogram body weight per day. With protein intakes ranging from 0.5 to 4.0 g. per kilogram of body weight per day, utilization of protein for tissue synthesis was also proportional to the protein intake. There was good agreement with published data on normal individuals as well as with observation on cancer patients and on patients, convalescing from gastrectomies

for peptic ulcers, fed through oral jejunal tubes. The observations of Benditt and his associates are not in accord with those of A. Keys *(J. Am. Dietet. A.* 22: 582, 1947) who stated that in relief feeding, calories are of overwhelming importance. Benditt *et al.* conclude that while caloric intake is a factor, it is not the only factor. Moreover, with an adequate caloric intake, the level of protein intake and the quality of the protein become the limiting factors. Protein intake levels of 2 to 4 g. per kilogram per day are demanded for rapid *rehabilitation* of a depleted person. This in contrast to the 1 g. per kilogram allowance for *maintenance* of a normal adult as stated by the National Research Council.

Benditt and his co-workers suggest that the rational procedure is to feed protein as a source of building material and compute the energy value of the diet, independent of its protein content, on the basis of its carbohydrate and fat content. For an average sized man (surface area 1.7 square meters afebrile and at bed rest, 2600 calories per day or 1500 calories per square meter per day are needed.

The two papers by Benditt *et al.* provide convincing summaries to show the value of high protein intakes during rehabilitation following protein depletion. Nevertheless the protein need is closely related to the caloric intake. In the rat experiments providing constant protein intake with variable caloric intakes, the level of protein fed was not high, and at maximum utilization the diet provided about 15 per cent of protein. Much better utilization was obtained upon a diet providing only 11 per cent of protein when food intake was higher. With additional experiments it should be possible to show the level of protein intake which will yield maximum efficiency for various levels of caloric intake.

Nutritional Status and Infection Response

Condensed from *Nutrition Reviews*, 6:205, July, 1948

WITH the aim of gaining information concerning the relation of nutritional status to the response to infection, studies have been initiated by J. Metcoff and associates *(J. Lab. Clin. Med.* 33:47, 1948). They describe the host hematopoietic, immunologic, and plasma protein responses to infection with Salmonella typhimurium, as these responses were influenced by two contrasting levels of protein intake.

Carefully planned and well-controlled studies were carried out with 60 rats, half of which received an adequate diet containing 18 per cent casein while the remainder were fed a diet containing 2 per cent casein.

Total circulating plasma proteins, particularly the gamma globulins, were relatively uninfluenced by infection per se in both well-nourished and protein deficient animals despite observed changes attending protein deficiency. With protracted protein deficiency, the circulating plasma proteins per unit of surface area were decreased. Infection was associated with slight reduction in the animals fed adequate protein, as compared with the controls, but both groups of deficient animals had similar unit circulating protein levels. The decreased unit

circulating plasma proteins appeared to be due to a depletion of albumin and gamma globulin components, independent of infection.

In the presence of infection there was decreased circulating hemoglobin per unit of surface area both in the well-nourished and in deficient infected rats. The total leucocyte counts of the deficient groups were somewhat lower than those of the well-nourished rats. The initial post-infection relative lymphopenia manifested by both infected groups was similar, however. Protein deficiency was associated with a tendancy toward a relative increase of immature myeloid forms in the femoral bone marrow and decreased numbers of maturing erythroid elements. Well-nourished infected animals appeared to have a relatively greater number of maturing erythroid forms than the controls. Dietary deficiency appeared to influence the bone marrow more than infection.

No significant difference in the incidence of bacteremia was noted between infected rats on the 2 per cent or the 18 per cent casein diets. Despite relative quantitative depletion of circulating gamma globulin, protein deficient rats attained antibody titers equivalent to those of well-nourished animals in response to infection. Thus, bacteriologic and immune responses resulting from infection were essentially unaltered by severe dietary protein deficiency.

These observations inject a note of caution, long overdue, in regard to the alleged relation between hunger, famine, and infection. In recent years, protein nutrition has acquired considerable prominence in this regard. The experimental studies of P. R. Cannon *(Advances in Protein Chemistry* 2:35, 1945) suggest that there is an increased susceptibility or a decreased resistance to infectious disease in protein inanition. Other investigators, as well as several clinical observers, have offered evidence to support this hypothesis. In general, laboratory studies pertaining to response to infection associated with protein deficiency have measured phagocytic indices, humoral antibody titers produced by a nonpathogenic antigen, or a lethal end point produced by a virulent, viable pathogen. There have been few concomitant studies of variations in specific physiologic responses to diet and infection. Since under any imposed condition the response of an organism to infection can be expected to be characterized by many adaptive phenomena, such studies are desirable before the implied importance of nutritional deficiency in relation to response to infection can be accepted as significant from a clinical standpoint. Metcoff *et al. (loc. cit.)* point out that the simple statement that protein deficiency leads to inadequate response to infection is an oversimplification of a many-faceted phenomenon. An appropriate experimental procedure involving the single variable of protein deficiency, they say, has not yet been reported. The genetic status of the host and pathogen, host factors initiating antibacterial immunity, the source of protein, protein deficiency conditioning other nutritional inadequacies as, for example, that of niacin, and numerous other fundamental variables must be simultaneously controlled before conclusions can be drawn. The importance of endocrine-protein metabolic interrelations and coincident nitrogen balance, liver protein, and protein synthesis studies should be helpful in interpreting the place of protein in infection response.

BOOK REVIEW

Oral Surgery. With 1631 illustrations including 121 in color. Two Volumes. *By* Kurt H. Thoma, D.M.D., Professor of Oral Surgery and Brackett Professor of Oral Pathology, Harvard University. Oral Surgeon and Chief of Dental Department, Massachusetts General Hospital. St. Louis: The C. V. Mosby Company, 1948. 1521 pp.

Dr. Thoma in the Preface of his new work, *Oral Surgery*, defines this specialty of dentistry as, *"a specialty encompassing all of the surgical diseases, injuries, and developmental abnormalities of the oral cavity, teeth, jaws and adjacent structures".*

To one who reads such a definition with thoughtfulness and understanding, the gargantuan proportions of the subject matter of a book covering so tremendous a field, is at once awesome. This may be particularly true with the general dentist or the student who will find a need for information in this field.

Without a doubt Dr. Thoma understood this fact well and has chosen to set down this treatise in the form of two Volumes. This was wise because neither one is too cumbersome to handle with ease while reading, nor so voluminous that one who is a frequent user of the text could not easily find his way through either book.

Volume I contains in its first two Chapters that information which it seems to this reviewer should act as a *stinging restraint* to the doctor who aspires to practice surgery without a proper background; at the same time, however, the knowledge contained in these two Chapters should furnish a treasure to be profoundly sought after by every student of oral surgery. These two Chapters deal with "The Principles of Surgery" and "The General Care of the Patient". One who well understands these two phases of practice will proceed with *safety*. They are so well written and well founded, as well as timely, that it would behoove every dentist and dental student to read them.

Volume I continues then in good sequence with the third Chapter covering "Dentoalveolar Surgery". This third Chapter should be of such vital interest to the general dental practitioner that it will stand in good stead to mention its contents briefly, and to clarify the title of the Chapter. "Exodontia"—some of us might term its contents! There is more to it than that however, and it is quite appropriate too that in a new book, *and this book is new*, a fitting name be given to the surgery of the alveolar ridges including the removal of teeth. Chapter III, "Dentoalveolar Surgery," includes what some have chosen to call "Minor Oral Surgery and Exodontia" and it is complete.

Dr. Celia Rich, with whom it is my pleasure to co-write this review, discusses the portion of Chapter III which deals with the surgery involving the gingivae in periodontal disease.

* * * *

At first glance, the periodontist will be disappointed when he finds less than twenty pages of this extensive work devoted to periodontal surgery, exclusive of periapical conditions. A brief investigation of these pages will convince him that Thoma has covered the field of radical procedures for periodontal pathology with the same scientific skill and thoroughness that characterize the balance of his subject matter.

It is understandable that in such a comprehensive study as his new *Oral Surgery*, Thoma has seen fit to leave to more specialized writings the general discussion of periodontal disease, including the type of surgery known as sub-gingival curettage.

Three types of procedure are described in detail: the gingival flap operation, electrocoagulation, and gingivectomy, and with each the special indications—general preparation of the patient, type of anaesthesia, and operative preparation of the mouth. The necessary instruments are described, as well as post-operative care and treatment of complications.

Three extremely interesting case histories, successfully treated by gingivectomy, are presented; one of gingival hypertrophy with pregnancy tumors, another of dilantin hypertrophy and a case of hypertrophy with excessive bleeding (accredited to Dr. Henry M. Goldman). Too much cannot be said of the illustrations which clarify the text.

Chapters I and II of *Oral Surgery* dealing with the general principles of surgery and with the general care of the patient respectively, and especially pages 368 and 386 of Chapter III, briefly described above, constitute "must" reading for every periodontist.—[C. R.]

The relatively new and astonishingly graphic two-color sketches by Miss Etta Piotti of various surgical procedures in these volumes should prove most helpful to the student, as well as to the more learned, who aspires to improve his technique. The photography is excellent but not over-done and is a valuable visual aid. The roentgenograms have been well reproduced by the printer and their usefulness as an adjunct to good treatment is not minimized by the author.

Anatomy is dealt with as a part of the description and graphic presentation of the actual cases. The complete case reports which are numerous in both Volumes will prove most helpful.

Fractures of the mandible are quite thoroughly covered, but the care of fractures of the maxilla and associated bones of the face are not as completely discussed as in some other texts.

Tumors, both benign and malignant, are frequently introduced and complete case reports of such a great number are invaluable. The significance of the pathological examination of all neoplasms removed from the mouth is emphasized. Far too many valuable biopsy specimens have haplessly found their way to the waste receptacle in the past, with no attempt being made to determine their minute structure.

The physician and general surgeon will find Thoma's Volumes of inestimable value. They are perhaps the first books dealing with the subject of Oral Surgery that can be readily understood by the general surgeon and also be found useful to him. They should be on the shelves of all medical libraries. Many problems in oral surgery, because of economics, will be dealt with by the general surgeon. The lack of surgical training in the general dental practitioner will make this necessary in small communities for a long time.

These Volumes give the most complete and authentic coverage of the subject of oral surgery that the profession has had at its disposal up to this time and it is sincerely hoped that copies will find their way into the hands of not only the oral surgeon, general dentist, and general surgeon, but into the hands of the dental and medical student.

—C. J. Speas

American Academy of Dental Medicine

The American Academy of Dental Medicine will hold its Annual Mid-Winter Meeting and Luncheon at the Hotel Pennsylvania, on Sunday, December 5th, 1948, at 12:30 P.M. Round Table discussions on dental-medical subjects will follow. All members and interested dentists and physicians are invited. Address to Dr. Wm. M. Greenhut, National Secretary, 124 East 84th St., New York 28, N. Y.

Attention! Academy Members

Plan now to attend the Post-Graduate Seminar, American Academy of Periodontology, W. K. Kellogg Foundation Institute, University of Michigan, Ann Arbor, Michigan—May 2, 3, 4, 1949. Harold G. Ray, *Chairman*

The Ohio State University Post-Graduate Courses In Dentistry

The College of Dentistry of The Ohio State University is offering another series of post-graduate courses for dentists and dental hygienists for the Winter Quarter of 1949. Every effort will be made to give each applicant the course or courses desired, but, because each group is limited to ten students, this is not possible in all instances. For this reason we suggest that you apply early and give an alternate choice.

The following courses will be offered in both the Winter and Spring Quarters:

X-ray; Anatomy; Periodontia; Full Dentures; Partial Dentures; Oral Surgery; Oral Pathology.

The following courses will be offered for one quarter only:

Winter Quarter, 1949—Dental Caries Control; Oral Hygiene for dental hygienists.

Spring Quarter, 1949—Crown and Bridge; Children's Dentistry; Operative Dentistry.

Schedule of Courses for Winter Quarter, 1949

X-ray (Dr. Spangenberg) Jan. 10-14
Oral Pathology (Dr. Robinson) Jan. 17-21
Partial Dentures (Dr. Steffel) Jan. 24-28
Periodontia (Dr. Wilson) Feb. 14-28
Full Dentures (Dr. Boucher) Feb. 21-25
Oral Surgery (Dr. Snyder) Feb. 28-March 4
Dental Caries Control (The Staff) March 7-11
Anatomy of Head and Neck (Dr. Edwards) March 21-25

Each course is given for five consecutive days, Monday through Friday. The charge for each course is $50 with the exception of Oral Surgery which is $100. All of these courses have been approved by the Veterans Administration, and veterans may have their fees paid under the provisions of the G.I. Bill of Rights if they present their Certificates of Eligibility when registering for the first time. Civilian dentists are required to send in a deposit of $25 with their applications.

Further information and application may be obtained from the Post-Graduate Division of the College of Dentistry, Ohio State University, Columbus 10, Ohio.

A course in Oral Hygiene for dental hygienists will be given from January 31 to February 4. The fee for this course is $25.

Schedule of Courses for Spring Quarter, 1949

Periodontia (Dr. Wilson) March 28-April 1
Oral Pathology (Dr. Robinson) April 4-8
X-ray (Dr. Spangenberg) April 11-15
Partial Dentures (Dr. Steffel) April 25-29
Children's Dentistry (Dr. Pettit) May 2-6
Full Dentures (Dr. Boucher) May 9-13
Crown and Bridge (Dr. Starr) May 16-20
Oral Surgery (Dr. Snyder) May 23-27
Anatomy (Dr. Edwards) June 13-17

University of Illinois News

Two separate postgraduate courses in "Recent Advances in Caries Control" and "Periodontia III: Histopathology of the Gingival and Periodontal Structures" will be offered on February 11-12 by the University of Illinois College of Dentistry.

The courses will be conducted on a Friday and Saturday, immediately following the mid-winter meeting of the Chicago Dental Society.

Dr. Robert G. Kesel will direct the course in "Recent Advances in Caries Control." Dr. Balint Orban is in charge of the second course, "Periodontia III," which includes microscopic study.

Enrollment in the course is limited. Registration may be made by writing to Dr. Isaac Schour, University of Illinois College of Dentistry, 808 S. Wood St., Chicago 12, Ill. The courses will be offered under the provisions of the G. I. Bill of Rights, but are open to non-veterans as well.

Back Copies Needed!

Any reader having in his possession the following numbers and wishing to part with them will please communicate with the Editor, 555 W. Maple Avenue, Birmingham, Michigan.

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JANUARY 1933

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JANUARY 1943

JANUARY 1944

JANUARY 1945

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